International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Jamwal S et al. Int J Reprod Contracept Obstet Gynecol. 2025 Oct;14(10):3559-3563
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20253109

Case Report

A rare case of postpartum cerebral venous thrombosis and hemmorhagic infarct

Sonal Jamwal[1]*, Drishti Kaushal[2], Arushi Rattan[2]*

[1]Department of Obstetrics and Gynecology, Civil Hospital, Ghawandal, Bilaspur, Himachal Pradesh, India
[2]Department of Opthalmology, IGMC Shimla, Himachal Pradesh, India

Received: 28 May 2025
Revised: 06 July 2025
Accepted: 7 July 2025

***Correspondence:**
Dr. Arushi Rattan,
E-mail: drsonal0225@gmail.com

ABSTRACT

Cerebral venous thrombosis (CVT) is a rare but serious complication that can occur during pregnancy and the postpartum period. This case reports discusses a unique instance of postpartum CVT complicated by hemorrhagic infarct in a patient from Himachal Pradesh. A case of a 26-year-old woman, para 2, unbooked patient with normal vaginal delivery at home, presented on 10th postpartum day with complaint of vomiting, headache, left hemiplegia and seizures, initially misdiagnosed as eclampsia, underwent CT, revealed significant sigmoid sinus thrombosis with haemorrhagic infarction. The patient underwent left fronto-temporoparietal decompressive hemicranectomy with duroplasty at PGIMER Chandigarh followed by tracheostomy. The patient was under antibiotic treatment, seizure prophylaxis, anticoagulation therapy, resulting in substantial clinical improvement. This case emphasizes the necessity of maintaining a high index of suspicion for CVT in postpartum women displaying neurological symptoms. Misdiagnosis can impede timely intervention, highlighting the need for access to advanced imaging and multidisciplinary approach. Increasing awareness and training for healthcare providers are essential for timely diagnosis. Also, the importance of hospital delivery. The management of this case illustrates the importance of regular antenatal checkup, hospital delivery, early recognition of CVT in the postpartum population. Enhancing diagnostic resources and treatment strategies, particularly in resource limited settings, can significantly improve maternal health outcomes and reduce morbidity and mortality. The possibility of cerebral vein thrombosis should be considered in all women with brain dysfunction during the puerperium.

Keywords: Cerebral venous sinus thrombosis, Postpartum, Haemorrhagic infarction, Eclampsia, Maternal health, Anticoagulation

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare but serious cause of stroke, with an increased incidence during pregnancy and the postpartum period.[1,2] However, recent study from a hospital in South India reported an incidence of 3.9 per 1,000 obstetric admissions.[3]

CVT commonly involves the superior sagittal and transverse sinuses, which are significant sites for thrombosis in affected individuals.[4-9] A significant challenge in the management of postpartum CVT is its frequent misdiagnosis as preeclampsia or eclampsia due to overlapping clinical features. Several risk factors contribute to the development of CVT in these populations, which are important to consider for early identification and management. Pregnancy induces a hypercoagulable state due to hormonal changes, which increases the risk of thrombus formation. This state persists for several weeks postpartum.[10] Women who undergo caesarean sections are at a higher risk for CVT, likely due to surgical trauma and the associated

inflammatory response.[11] This pregnancy complication is associated with increased risk factors for thrombosis, including elevated blood pressure and proteinuria, which can contribute to CVT.[11] Infections during pregnancy or the postpartum period can exacerbate the risk of thrombosis, as they may lead to dehydration and other physiological changes that promote clot formation.[12] Conditions such as anaemia and elevated platelet counts during pregnancy can increase susceptibility to CVT. This can occur during labor and delivery, particularly in cases of significant blood loss, further increasing the risk of CVT. Additional risk factors include obesity, smoking, and a history of thrombophilia, which can further complicate the clinical picture.[12] This diagnostic ambiguity can lead to delays in appropriate treatment and potentially adverse outcomes. Prompt diagnosis, facilitated by neuroimaging, is crucial to prevent life-threatening complications. This case report describes a unique instance of postpartum CVT initially misdiagnosed as impending eclampsia, highlighting these diagnostic challenges and emphasizing the importance of timely intervention in a resource-limited setting.

CASE REPORT

A 26-year-old woman, para 2, presented to the hospital on the 10th postpartum day following vaginal delivery conducted at home, attended by a traditional birth attendant. The patient exhibited a 1-day history of left-sided weakness and generalized tonic-clonic seizures. Prior to the onset of her neurological manifestations, the patient experienced an abrupt, severe generalized headache accompanied by blurred vision and visual field deficits. Additionally, her attendants reported rapid mood fluctuations and initially sought care at a local clinic, where she received unspecified oral medication without any improvement, but was unable to provide further details regarding the type or composition of the medication. Considering the patient's condition, a thorough exploration of her obstetric history was undertaken to identify any previous complications associated with childbirth, particularly venous thromboembolism (VTE) or indications for thrombophilia screening. The patient attendant denied any prior history of postpartum complications or VTE, and her medical records revealed no documentation of thrombophilia screening. This absence of previous events may have contributed to the initial oversight of her symptoms during the current postpartum period.

Upon admission to the gynaecology ward of a civil hospital in Ghawandal, Bilaspur, the patient was administered diazepam; however, her level of consciousness subsequently declined. The patients past medical history were unremarkable, with no known hypertension, diabetes, epilepsy, or history of oral contraceptive use. She denied any known drug or food allergies. The patient was married, had two children, resided in traditional housing, and reported no history of substance abuse.

Physical examination

On admission, the patient exhibited a reduced level of consciousness with a Glasgow Coma scale (GCS) score of 9/15. She appeared restless and agitated, was normocephalic without facial asymmetry, but exhibited neck stiffness. Vital signs included blood pressure of 130/90 mmHg, heart rate of 100 bpm, respiratory rate of 20 /min, oxygen saturation of 96% on room air, and temperature of 36.9 °C. Neurological examination revealed vigorous movements of the right limbs, left-sided hypotonia, and an equivocal plantar response.

Investigations

Complete blood count (CBC) was total WBC 11,100 cells/µl; (neutrophils 9500 cells/µl), hemoglobin 7.5 g/dl, MCV 61.2%, platelets 455,000 cells/µl. Renal function tests (RFT) was urea 17 mg/dl, creatinine 0.6 mg/dl. Liver function tests (LFT) was GOT 17 IU/l, GPT 21 IU/l, total bilirubin 0.8 mg/dl. Blood group was O positive. Urine analysis: pH 5, protein 2+, WBCs 1-5 /HPF. Following history for presumed eclampsia and postpartum psychosis, a brain CT scan was performed, revealing an irregularly shaped hyperdense hemorrhage in the left frontal lobe at the gray white matter junction, accompanied by surrounding hypodense edema and effacement of the ipsilateral lateral ventricle, indicating mild midline shift to the left (Figure 1).

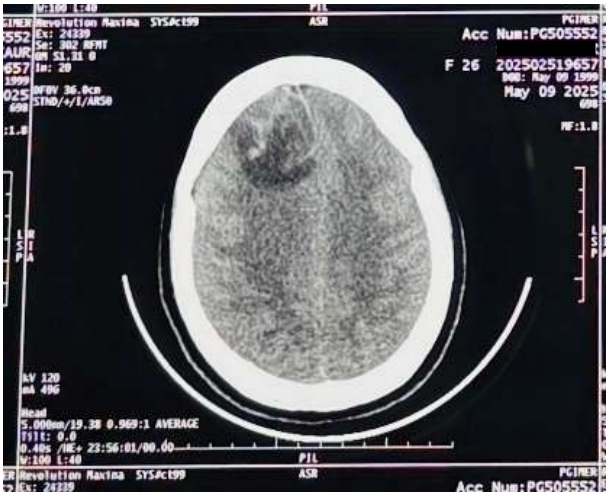

Figure 1: Axial non contrast brain CT scan shows irregularly shaped hyper dense hemorrhage in the left frontal lobe on at grey white matter junction with surrounding hypo dense edema.

The findings suggested a large venous hemorrhagic infarction secondary to superior sagittal sinus thrombosis, complicated by mild subarachnoid hemorrhage, brain edema, and mild subfalcine herniation to the left.

Patient referred to higher centre where CT cerebral venography was done which confirmed superior saggital

sinus thrombosis, left transverse and SSS with left frontal hemmorhagic infarcts (Figure 2).

Treatment/management provided

Surgery done was left fronto-temporoparietal decompressive hemicraniectomy with lineae drill and bifrontal approach by neurosurgery team. Bone flap was placed in abdomen in one piece. ASD was done. Tracheostomy was done next day due to prolonged ventilation and shifted to medicine side with seizure prophylaxis of phenytoin 100 mg PO TID. For anticoagulation, unfractionated heparin (UFH) 5000 IU IV, injection piperacillin tazobactum 12 hourly, injection vancomycin 8 hourly were given.

Other medications were paracetamol 300 mg IV as needed, intravenous normal saline 1000ml BID, and bromocriptine 2.5 mg PO BID.

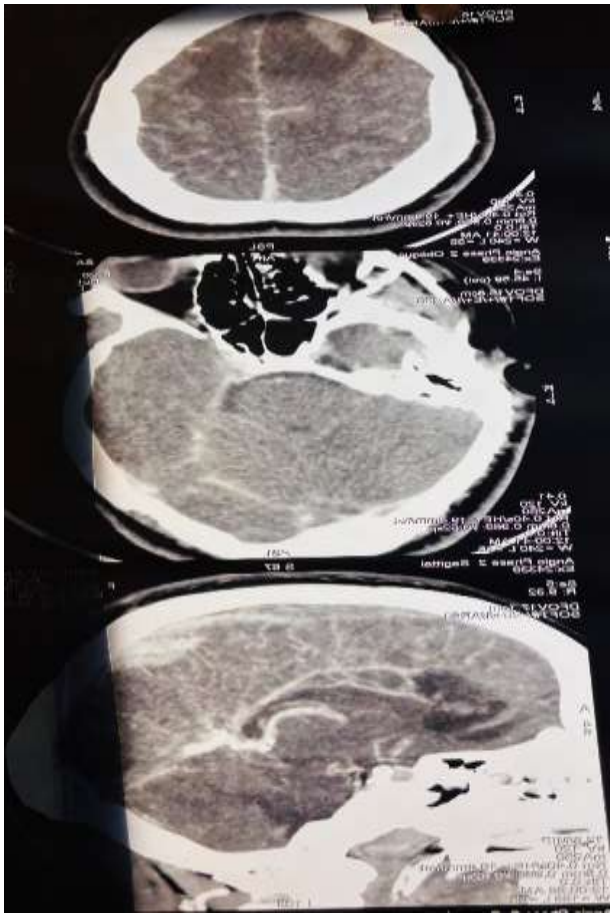

Figure 2: CT venography, absent flow signal in superior sagittal sinus and left transverse sinus.

Clinical course and follow up

By day 3 of admission, the patient GCS is E2VTM5 on tracheostomy with tracheostomy site clear. Coagulation studies indicated a prothrombin time (PT) of 24 seconds, partial thromboplastin time (PTT) of 27.5 seconds, and an INR of 2.4. On day 9, her GCS improved to E4VTM6, although she retained left hemiplegia (power 0/5). The INR was noted to be 3. She was discharged on day 16 with tracheostomy removed, residual word-finding difficulties and flickering movements in her left hand. Warfarin therapy was continued at 2.5 mg daily. At a one-month follow-up, the patient was able to walk with slight support, exhibiting power of 4/5 and continued word-finding difficulties, along with sleep disturbances.

DISCUSSION

We reported a case of postpartum CVT in a 26-year-old woman from Himachal Pradesh, complicated by haemorrhagic infarction. The patient's initial symptoms were severe headache, seizures, and focal deficits-closely resembled eclampsia, which led to a delay in accurate diagnosis.

However, prompt recognition of CVT through CT and CT venography, followed by appropriate medical management, resulted in a positive clinical outcome. This case highlighted the diagnostic challenges of CVT, particularly in settings where advanced imaging may not be immediately available, and emphasized the importance of maintaining a high level of suspicion for this condition in the postpartum period.

Cerebrovascular disorders, while uncommon, pose significant risks to both mother and child during pregnancy and the postpartum period, potentially leading to life-threatening and disabling complications. They can be divided into two main categories: Thrombosis and ischemia, which include arterial and venous strokes and hemorrhage.[4,5,13,14]

Furthermore, Adam et al noted that a significant proportion (56.9%) of patients with CVT in their study had no identifiable pre-existing risk factors, emphasizing the need for heightened clinical suspicion for CVT even in the absence of traditional risk factors in this population. The underlying pathophysiology involves a complex interplay of hypercoagulability, venous stasis and endothelial dysfunction, all of which are heightened during pregnancy and the postpartum period.[18]

Several factors contribute to the increased risk of CVT in the postpartum period, including hypercoagulability related to pregnancy, caesarean delivery, infections, blood loss during delivery and dehydration, fluctuations of intracranial pressure during labor, hypertensive complications of pregnancy, and even cerebrospinal fluid loss following dural puncture.

Consistent with previous studies, our case underscores the postpartum period as a significant risk factor for CVT.[1,2] The patient's clinical presentation-headache, visual changes, seizures, and neurological deficits aligned with common manifestations of CVT.[2,13,16-18] The potential contribution of dehydration, possible infection, and

especially anemia to the patient's hypercoagulable state cannot be definitively ruled out given the limitations in available documentation and diagnostic resources. Anemia itself is a known risk factor for thrombosis during pregnancy and postpartum.[17,21] While the WBC count of 11,100 cells/µl suggested possible infection, further investigation was not possible. The absence of formal evaluation for these conditions underscores the challenges of providing comprehensive maternal care in resource-limited settings and highlights an area for future improvement. These circumstances underscore the need for enhanced resources and trained healthcare professionals to prevent, identify, and manage modifiable risk factors for CVT.

A key challenge in this case was the initial misdiagnosis of eclampsia, a common condition in postpartum women with similar symptoms. This underscores the documented difficulty in differentiating between CVT, eclampsia, and other conditions such as posterior reversible encephalopathy syndrome (PRES).

The patient's initial clinical picture led to treatment for eclampsia, further emphasizing the need to consider fewer common etiologies when patients do not respond to conventional treatment.

A significant diagnostic challenge in this case, and in others like it, is the considerable overlap in clinical features between CVT and preeclampsia. Both conditions frequently present with severe headaches, seizures, visual disturbances, and altered mental status, particularly in the postpartum period. Moreover, the shared risk factor of the hypercoagulable state associated with pregnancy and the puerperium further complicates the diagnostic process.[22-25] In both conditions, endothelial dysfunction & inflammatory process may contribute to cerebral edema and neurological symptoms.[15,25] This convergence of clinical and pathophysiological features makes differentiating between CVT and preeclampsia a significant challenge, demanding a high index of suspicion and the judicious use of neuroimaging.

This case adds to the existing literature by highlighting several key points. First, the occurrence in a low-resource setting in Ghawandal, Bilaspur, where access to advanced imaging and prompt specialized care may be limited, emphasizes the need for heightened clinical vigilance and the use of CT scan as an acceptable initial imaging method. Second, the patient was unbooked, not taken iron folic acid tablet and also delivery was conducted at home recognized risk factors for CVT. Third, the patient was managed by a multidisciplinary team, which resulted in a positive outcome. Lastly, this case highlights the importance of multidisciplinary care for CVST patients.

This case highlighted significant challenges in the diagnosis and management of maternal stroke and underscores the urgent need for targeted policy interventions.

To enhance early recognition and improve outcomes for affected women, we recommend the following actions.

Healthcare provider training enhancement

Integrate stroke education, particularly regarding atypical presentations during the postpartum period, into existing maternal health training programs. Focus this training on frontline healthcare providers working in primary care, antenatal clinics, and emergency obstetric settings to promote early identification and timely referrals.

Culturally sensitive public awareness campaigns

Launch educational initiatives aimed at pregnant and postpartum women, highlighting common and atypical signs of stroke such as persistent headache, visual disturbances, seizures, and focal neurological symptoms. Educational materials should be made available in multiple local languages and disseminated through diverse formats, including posters, brochures, and radio messages. These campaigns must emphasize the urgency of seeking immediate medical care when symptoms occur, particularly in the postpartum period. Also, every delivery to be conducted at hospital.

Improved referral and transport systems

Strengthen the healthcare referral infrastructure to ensure timely transfer of women with suspected strokes to facilities equipped with diagnostic tools (e.g., CT or MRI scanners) and neurological expertise. This includes establishing clear referral protocols, enhancing communication between facilities, and ensuring reliable transportation options.

Data collection and surveillance systems

Establish a surveillance system to collect data on maternal strokes, including incidence, associated risk factors, and patient outcomes. This data will be vital for monitoring trends, evaluating interventions, and informing future policy decisions.

Strategic resource allocation

Advocate for increased investment in maternal health services, specifically targeting resources for stroke-related care. This should include funding for educational programs, diagnostic imaging equipment, and access to specialist neurological services.

CONCLUSION

This case illustrates a rare but serious occurrence of postpartum CVT with haemorrhagic infarction in a 26-year-old woman from Ghawandal. The case emphasizes the urgent need for increased awareness and early recognition of CVT in postpartum patients, particularly when they present without typical risk factors or

symptoms. The diagnostic delay due to limited resources and atypical presentation highlights the importance of improving access to neuroimaging and training healthcare providers to consider CVT in differential diagnoses. Enhanced diagnostic capacity and timely intervention are essential to reduce complications, improve outcomes, and save lives in similar settings.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Singh A, Singh S, Vardhan S. A rare case of post-partum cerebral venous sinus thrombosis. Int J Reprod Contracept Obstet Gynecol. 2018;7(2):762.
2. Jain S, Bhushan M, Talwar V. Postpartum cerebral venous sinus thrombosis: a case report. Qatar Med J. 2024;2024(1):63-7.
3. Sasikala R, Aishwarya J, Dilshath S. Clinical profile of cerebral venous thrombosis in pregnancy and puerperium in South India. Int J Reprod Contracept Obstet Gynecol. 2017;6:3124-8.
4. Capecchi M, Abbattista M, Martinelli I. Cerebral venous sinus thrombosis. J Thromb Haemost. 2018;16(10):1918-31.
5. Agrawal K, Burger K, Rothrock JF. Cerebral sinus thrombosis. Headache J Head Face Pain. 2016;56(8):1380-9.
6. Alvis-Miranda HR, Castellar-Leones SM, Alcala-Cerra G, Moscote-Salazar LR. Cerebral sinus venous thrombosis. J Neurosci Rural Pract. 2013;4(4):427.
7. Gazioglu S, Dinc G. Cerebral venous sinus thrombosis in pregnancy and puerperium. Acta Neurol Belg. 2021;121(4):967-72.
8. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis: a cross-sectional study. Stroke. 2012;43(12):3375-7.
9. Saposnik G, Bushnell C, Coutinho JM. Diagnosis and management of cerebral venous thrombosis: a scientific statement from the American Heart Association. Stroke. 2024;55(3):77-90.
10. Silvis SM, Lindgren E, Hiltunen S. Postpartum period is a risk factor for cerebral venous thrombosis. Stroke. 2019;50(2):501-3.
11. Bajko Z, Motataianu A, Stoian A. Postpartum cerebral venous thrombosis-a single-center experience. Brain Sci. 2021;11(3):1-9.
12. Wang S, Yao M, Hu N, Liu J, Peng B. The risk profiles of pregnancy-related cerebral venous thrombosis: a retrospective study in a comprehensive hospital. BMC Neurol. 2024;24(1):1-11.
13. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. Lancet Neurol. 2007;6(2):162-70.
14. Schaller B, Graf R. Cerebral venous infarction: the pathophysiological concept. Cerebrovasc Dis. 2004;18(3):179-88.
15. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke. 2004;35(3):664-70.
16. Grossberg AJ, Galwankar SC. Cerebral venous thrombosis in pregnant and postpartum women presenting to the emergency department with headaches. J Emerg Trauma Shock. 2024;17(1):3-7.
17. Wang N, Shen X, Zhang G, Gao B, Lerner A. Cerebrovascular disease in pregnancy and puerperium: perspectives from neuroradiologists. Quant Imaging Med Surg. 2021;11(2):838-51.
18. Sidorov EV, Feng W, Caplan LR. Stroke in pregnant and postpartum women. Expert Rev Cardiovasc Ther. 2011;9(9):1235-47.
19. Mas JL, Lamy C. Stroke in pregnancy and the puerperium. Neurol. 1998;245(6):305-13.
20. Liberman AL, Gialdini G, Bakradze E, Chatterjee A, Kamel H, Merkler AE. Misdiagnosis of cerebral vein thrombosis in the emergency department. Stroke. 2018;49(6):1504-6.
21. Moumin HE, Ali AO, Egge AAA, Abdi MH, Moumin AE, Muse AH. Spontaneous splenic rupture: a rare complication of concurrent malaria and dengue infections-a case report. Int J Surg Case Rep. 2025;126:110783.
22. Agarwal M, Hughes P, Ezimokhai M. Maternal fetal medicine. Obs Gynecol. 2000;182(346):50.
23. Olomu SA, Olaniyi JA, Shokunbi WA. Coagulation profile according to gestational age in pregnant Nigerian women. Trop J Obstet Gynaecol. 2007;24(1):3-6.
24. Olomu SA, Olaniyi JA, Shokunbi WA. 55 coagulation profile according to gestational age in pregnant Nigerian women. Thromb Res. 2007;119:112.
25. Burton GJ, Redman CW, Roberts JM, Moffett A. Pre-eclampsia: pathophysiology and clinical implications. BMJ. 2019:366.

Cite this article as: Jamwal S, Kaushal D, Rattan A. A rare case of postpartum cerebral venous thrombosis and hemmorhagic infarct. Int J Reprod Contracept Obstet Gynecol 2025;14:3559-63.